DISORDERS
OF
METABOLISM
AND
NUTRITION

VON NOORDEN

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By Prof. Dr. Carl von Noorden
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CLINICAL TREATISES
on the
PATHOLOGY *and* THERAPY *of* DISORDERS *of* METABOLISM *and* NUTRITION

BY

PROF. DR. CARL VON NOORDEN

Professor of the First Medical Clinic, Vienna

PART VIII
INANITION AND FATTENING CURES

AUTHORIZED AMERICAN EDITION

Edited and translated under the supervision of

ALFRED C. CROFTAN, M. D.

Chicago, Ill.

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NOTE BY THE AMERICAN EDITOR

The phenomena of undernutrition occurring either independently or as a complication of a variety of diseases are often overlooked, or if discovered at all, are commonly misinterpreted. Hence chronic undernutrition is frequently allowed to persist to the great detriment of the organism. Three fundamental rules must be fulfilled in feeding patients, especially individuals afflicted with chronic disorders, viz.: 1. The diet must incorporate a caloric value sufficient to maintain adequate nutrition, for otherwise the body cannot put forward its best endeavors to combat the disease. 2. The diet must be so composed that it spares the diseased organ, i. e., imposes no excessive tasks upon parts that are already functionally or organically impaired. 3. The diet must neither directly nor indirectly injure or overtax any portion of the body. Regulations both of a qualitative and a quantitative character, must therefore, be formulated; and these rules should be based upon physiologic reasoning, and only when this fails upon empiricism. Routine of any sort is to be condemned. The peculiar nutritional demands of each patient should be carefully studied. Accordingly, "not only must the patient be instructed by the physician, but the physician must also learn from each patient." Sanatoria and

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watering places should learn this lesson more thoroughly.

The rules in regard to the *quality* of the diet that patients suffering from different diseases should have are more easy to understand than the rules in regard to the total *quantity* of food that is permissible and advisable. There is a normal state of nutrition, a maximum and a minimum compatible with safety and well-being. Above and below these levels lies dangerous territory. In order to understand the regulation of these limits the mathematics of the normal, expressed in calories and energy equivalents, must be understood.

The two lectures published herewith do this in a masterful manner. The conception of the "maintenance diet" (*Erhaltungskost*) is formulated and made the basis of all nutritional studies; the effects of deviations from this standard (acutely or chronically—fasting or undernutrition) upon the fats, the carbohydrates, the proteids of the body are clearly and concisely delineated; the most important urinary phenomena that are the direct result of general undernutrition or of one-sided feeding are described and their diagnostic significance interpreted.

In the second lecture the treatment of chronic undernutrition is given, i. e., the raising of the state of nutrition from the sub-normal to the normal, or from the normal to the maximum. Especially instructive, from a practical standpoint, are the rules in regard to the increase of active, breathing, energy-producing muscle protoplasm on the one hand; of inactive, storage—or reserve—material, chiefly represented by fat, on the other. It is particularly refreshing, in this lecture, to read the criticism of innumerable dietetic fads and fallacies. Iconoclasm of this kind is of the greatest practical use provided something of value is erected in the place of the fallen idols. This requirement the author of these lectures has fulfilled to a pre-eminent degree.

ALFRED C. CROFTAN.

April 25th, 1910.
100 State Street, Chicago.

PREFACE TO THE AMERICAN EDITION

THE four lectures comprised in this monograph and in the succeeding one (Part IX, Reduction Cures and Gout) were delivered in a post-graduate course for Vienna physicians (May, 1908). By request of a number of local and foreign hearers, I am herewith publishing them in a somewhat extended form. I have omitted literature references throughout, as the latter may be found complete in my Handbook on the Pathology of Metabolism that has recently appeared (Berlin, 1906-1907). A few of the latest publications, however, that have been issued since then, are quoted. Whereas in my Handbook the facts that underlie our knowledge of the disorders of metabolism are exhaustively and critically explained, I have limited myself in these lectures essentially to a discussion of certain general conclusions that are based on theoretical and clinical experience and that are of value in actual practice. It was impossible, of course, to refrain altogether from occasional excursions into the theoretical side of the subject.

Two other lectures delivered in the post-graduate course of last year dealt with the Treatment of Diabetes Mellitus. I have omitted them from this series because everything essential contained in these lectures is incorporated in my book on "Diabetes Mellitus and its Treatment," 4th Edition, 1907, issued by the publishers of this Monograph.

CARL VON NOORDEN.

CONTENTS

PART I

INANITION AND UNDERNUTRITION

PAGE
I. Definition and Occurrence 13
II. Loss of Weight 18
III. Participation of the Different Organs in the Loss of Weight 21
IV. Combustion Processes 23
V. Distribution of Losses over Proteids, Fats and Carbohydrates 27
VI. The Excretion of Acetone Bodies . . 34
VII. Other Properties of the Urine during Inanition 41
(a) Ammonia 41
(b) Sodium Chloride 42
VIII. Numerical Data for Calculating the Degree of Undernutrition 44
IX. Composition and Caloric Value of Some of the Important Articles of Food . 47

PART II

FATTENING CURES

I. Definition of the State of Nutrition . 51
1. Muscular Tissues 51

PAGE
2. Adipose Tissues 54
II. CONDITIONS PRELIMINARY TO A FATTENING CURE 56
III. INCREASE OF MUSCULAR AND ADIPOSE TISSUE 59
1. Increase of Muscular Tissue . . . 59
2. Increase of Adipose Tissue . . . 70
IV. FATTENING FOODS 73
1. Proteids 74
2. Carbohydrates 77
3. Fats 84
4. Alcohol 90
V. REST AND EXERCISE 93
VI. INDICATIONS FOR FATTENING CURES . . 98
1. Pulmonary Tuberculosis 98
2. Basedow's Disease (Exophthalmic Goitre) 99
3. Nervous Dyspepsia 102

PART I

INANITION AND UNDERNUTRITION

PART I

The Perversions of Metabolism in Inanition and Undernutrition

I DEFINITION AND OCCURRENCE

It is important that practitioners should understand the changes of the metabolism that occur in complete and partial withdrawal of food; otherwise certain striking phenomena may be erroneously interpreted to signify specific consequences of disease, that in reality are due to insufficient nutriment or to one-sided feeding. A very convincing example of this error is furnished by the history of acetonuria. Formerly a variety of forms of acetonuria were described. Aside from the diabetic form, there was an acetonuria due to inanition, a febrile acetonuria, an enterogenous, cachectic, etc., acetonuria. As a matter of fact none of these forms, with the exception of diabetic acetonuria, are in any way directly dependent upon specific morbid processes mentioned above, but are altogether dependent upon undernutrition, that is, upon a reduction of the amount of food that patients afflicted with such disorders are necessarily able to take. Similar errors are recorded in regard to changes in the excretion of urinary chlorides. As an example I can quote the case of a young, poorly nourished girl suffering from chlorosis, who during the course of several weeks, on account of gastric hyperesthesia, took only from 1½ to 2 liters of milk daily. The urine contained a trace of albumin but no renal elements; this finding being, as is well known, of relatively frequent occurrence in chlorosis. The urine was abundant in quantity and of a low specific gravity and contained only 2 g of Cl Na in the 24 hours' volume. From this evidence the conclusion was drawn that there was a serious retention of chlorides attributable to nephritis, and presumably contracted kidney. As a matter of fact the low excretion of chlorides in the urine was attributable exclusively to the small amount of chlorine contained in the low diet the patient was getting; so that this was an altogether false alarm. For in order to prove my contention, I ordered the addition of 10 g of table salt to the diet, with the result that the kidneys were found to be fully equal to the task imposed upon them, the whole amount of sodium chloride added to the diet appearing almost quantitatively in the urine within 36 hours.

Examples of this kind could readily be multiplied.

It is important from a practical standpoint to distinguish between two forms of inanition, namely:

1. *Complete fasting,* that is, complete withdrawal of all food either in cases that at the beginning of this fasting period were taking ample food, or in cases that were taking an insufficient quantity of food for a long time before.

2. *Chronic undernutrition* witnessed in patients who for weeks or months, and sometimes even for years, had been taking an amount of food that was not sufficient to completely cover the demands of the organism for nourishment. The latter form is the more common and from a practical standpoint the more important of the two.

In practice we encounter inanition and undernutrition under many different conditions:

1. *As an undesired accompaniment* of numerous diseases; of severe inflammatory and traumatic lesions of the upper digestive tract rendering it impossible or almost impossible to swallow food; of many acute, grave diseases of the stomach and intestine (severe forms of gastritis, enteritis, dysentery, cholera-like diseases); of many serious fevers with psychoses, many nervous diseases (psychic disorders, severe tabetic crises, hysterical hyperemesis, etc.), also of advanced circulatory disturbances—in all of which the patients may absolutely refuse food. In these and similar disorders we encounter all stages of inanition from complete fasting to restrictions of the food intake of varying degrees. This factor frequently causes serious concern and renders the treatment of such patients worrisome. Of course one should never be too anxious in regard to the introduction of food in many of these patients, for the danger of underfeeding mentioned above may be exaggerated. If the disease is of such a character that the complete or the almost complete reduction of the food intake is apt to last

only for a few days, then serious danger will never accrue from this source.

Chronic undernutrition as a part phenomenon of certain diseases is much more common and much more troublesome. It almost constantly accompanies chronic diseases that run a febrile course, primarily tuberculosis, and it is also seen in nearly all diseases of the stomach and intestine. A large number of these patients are recruited from the ranks of the neuropaths. Only in a small minority of the cases can any objectively determinable reason be discovered for failure of these patients to eat, digest and assimilate an amount of food that would be necessary to maintain their nutrition. As a rule we will encounter sensations of distaste, unreasonable prejudices or fears in regard to the consequences to be expected from the ingestion of certain kinds of food or of any food; or, again, the composition of the diet may be inappropriate, so that the patient does not really obtain what is needed to fully nourish him. (Compare Lecture II, paragraphs on Indications for Fattening Cures.)

2. *As a therapeutic means,* we occasionally prescribe complete fasting (even in cases that could take some food) in serious disorders of the stomach and intestine (inflammation, ulceration and in threatened inflammation of the peritoneum). In nervous dyspepsia with great reduction of the food intake there is hardly a better means to stimulate the appetite and in this way to reaccustom the patient to the ingestion of a sufficient amount of food than complete fasting for a period of

days. Much more frequently, of course, chronic undernutrition is employed therapeutically in obesity. In all reduction cures this method must be employed. In gout too and in certain circulatory disturbances and in some diseases of the kidneys, undernutrition at least for a few weeks, may be of great value.

II LOSS OF WEIGHT

When a well-nourished adult individual begins to fast completely, he begins to lose from 800 to 1000 g in weight daily (that is about 1 to 1½% of his body weight). The daily losses remain more or less constant during the first eight days of fasting, then they become smaller. I will refrain from entering into a discussion of the conditions as they appear at this later period, because they are without significance in actual practice. I will only mention in passing that in continuous fasting death occurs when from 35 to 40% of the original weight has been sacrificed. The loss of weight in a fasting individual is more or less independent of the intake of water (by mouth, subcutaneously or by rectum). Generally speaking the demand for water is much smaller in an individual who is fasting than in a person who is taking even a small, although altogether inadequate, quantity of food. Persons who make an exhibition of continued fasting and who are allowed to take water in any desired quantity rarely drink more than 400 to 500 cc. a day. In individuals who are already very much reduced by chronic undernutrition and then suddenly undergo acute fasting, the absolute daily loss of weight is usually slighter, whereas the percentic losses of weight are just as high as those indicated above.

In chronic undernutrition the daily loss of weight

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varies greatly. Everything depends on the degree of undernutrition. Occasionally one witnesses even in continued undernutrition an arrest of the loss of weight, in fact, occasionally a transitory gain in weight. This phenomenon is dependent upon the water ingestion and the degree of water retention in the tissues. The retention of water may attain very considerable degrees before it becomes manifest by visible or tangible oedemas. Such a gain in weight may often give rise to false expectations in regard to improvement, unless the above conditions are carefully included in the calculation. A deceptive gain in weight or an arrest in the loss of weight is especially frequent in undernourished patients suffering from stomach or intestinal trouble, diabetes mellitus and insipidus, exophthalmic goitre and, of course, particularly in disorders of the heart and kidneys that are apt to produce oedematous accumulations. The total loss of weight may become very much greater in chronic undernutrition than ever in acute complete fasting. A reduction of the weight to 50 and 40% of the original weight is by no means a rare observation in cases of pulmonary tuberculosis, carcinoma of the oesophagus and of the stomach and diabetes mellitus. The greatest loss of weight that I have personally ever seen as the result of chronic undernutrition occurred in a woman of about the age of 40, weighing 120 kg. By voluntary undernutrition she reduced her weight within a year to 80 kg. She then became afflicted with pulmonary tuberculosis accompanied by serious loss of appetite. All attempts to adequately nourish her were in vain and, when she finally succumbed, six months after this time, she weighed only 46 kg; this represented a loss of 74 kg, that is, of 62% of her original weight, within a period of 18 months.

III PARTICIPATION OF THE DIFFERENT ORGANS IN THE LOSS OF WEIGHT

The different organs participate very unequally in the loss of weight. If fasting is carried on experimentally until death occurs (immaterial whether death is produced by acute complete fasting or chronic undernutrition) then

The nervous system only loses about 1 to 2% in weight.

The bones 10 to 15%.

The organs containing much albumin, as blood, glands, muscles, 40 to 50%.

The adipose tissues suffer the most, losing from 70 to 90% of their bulk.

As an evidence of the loss of bone tissue large quantities of calcium always appear in the urine during complete fasting. An individual living on an average mixed diet generally excretes more magnesium than calcium in the urine. During fasting this proportion is reversed; for dwindling bone tissues contain more calcium than magnesium. If the percentage of earthy alkalies contained in the feces is included in the calculation, then a relation between the two is found that exactly corresponds to the percentic composition of bone tissue. The total average loss of calcium during inanition amounts to 0.4 g, of magnesium about 0.2 g per diem. In chronic undernutrition bone material is also sacrificed. It appears that even prolonged rest in bed, that is, inactivity of the skeleton, may produce this result. If at the same time the individual is in a condition of chronic undernutrition, then this effect becomes still more apparent, so that there develops a permanently increased excretion of earthy alkalies as compared to the quantity of earthy alkalies ingested with the food. This phenomenon has been termed demineralization, and the doctrine has been advanced that this occurrence is characteristic of certain definite diseases, in particular of pulmonary tuberculosis. It appears, however, on careful analysis, that one is dealing merely with one of the results of inanition and of the inactivity of the skeletal tissues. Only in diabetes mellitus and in diseases of the bones proper (osteomalacia, bone tumors, etc.) do we witness a destruction of bone tissue independent of inanition. Determinations of the urinary calcium, that were considered to be of great importance several decades ago, are now considered to be altogether without value, unless the calcium intake is known and the calcium contents of the feces is included in the calculation.

IV COMBUSTION PROCESSES

The development of energy is not materially altered in acute inanition; the absolute amount of energy developed, it is true, becomes reduced (external conditions being equal) with the reduction of the weight. Calculated per kilogramme, however, the total energy development expressed in calories is only slightly reduced.

Tigerstedt in a man who fasted for five days found the following:

Day of fasting	Weight kg.	Total Caloric development	Caloric development per kg.
1	70	2229	33.1
2	66.7	2102	32.0
3	64.8	2024	31.2
4	64.0	1992	31.1
5	63.1	1971	31.2

In investigations of this kind the total caloric development is best calculated from the quantity of oxygen consumed. The technique of this method of investigation has been worked out so thoroughly that now-a-days such determinations can be performed with all necessary precision. If no carbohydrates are consumed, and this is the case in a fasting individual, then the consumption of one liter of oxygen demonstrates that 4.7 calories were developed.

Respiratory metabolism and the total development of heat, therefore, proceed exactly in the same manner in fasting as in complete nutrition. The only difference is this: that in inanition the necessary amount of energy is derived from the combustion of the body tissues proper, whereas under normal conditions the food constituents should be the source of this energy.

In undernutrition in which one part of the caloric demands is supplied by the food, another part by the sacrifice of body tissues, conditions are similar. This may be considered established at least for early periods of undernutrition. There remains to be discussed merely the question whether or not with sinking of the body weight and sinking of the albumin contents of the body, a sort of adaptation occurs; in the sense namely, that the total development of energy becomes correspondingly reduced. This would constitute a conservative process of economy. The existence of such relations becomes apparent when the total number of calories developed is calculated. Assuming that a well-nourished individual undergoes chronic undernutrition until he has lost 30% of his original body weight, then he will require 30% less food intake to maintain this reduced weight than he would have required to maintain his original weight. This relation becomes especially clear in studies performed on individuals undergoing climbing tests. A person weighing 100 kg climbing an elevation of 1000 m performs a labor of 100000 kgm; an individual weighing 50 kg mounting to the same elevation develops 50000 kgm. The amount of energy actually consumed is approximately three times this amount (300000 and 150000 kgm), for a human being is capable of transforming only one-third of the developed energy into external labor. In the first instance the rise to the elevation demanded the development of 706 calories, in the second of 353 calories (1 calorie=425 kgm).

The relative development of energy, i. e., the number of calories developed per kilogramme of body weight, remains approximately the same in adequate and inadequate nutrition, leaving aside certain unimportant exceptions that need not be discussed in this place. One can predict with almost mathematical accuracy what loss of weight will occur from a definite degree of undernutrition. We will assume that an individual weighing 70 kg requires 40 calories per kilo of body weight in order to maintain nutritive equilibrium. Taking into consideration his occupation and method of life, we will now assume that instead of getting these 2800 calories per diem, he is forced for some reason or other to live on a diet containing only 2400 calories. The expenditure per kilo remains the same; and he will therefore be forced to develop the deficit of 400 calories from his own tissue substances, and he will continue to emaciate until his weight has been reduced to about 60 kg. When this point has been reached the balance has been re-established between the calories introduced with his food (2400 calories) and the calories that he must develop according to his body weight to maintain nutritive equilibrium. So long as his diet and the external conditions of life remain the same, he will not emaciate on this caloric intake.

This form of calculation applies to the great majority of cases. Only in extreme degrees of emaciation do exceptions occur, for here a greater amount of energy must be developed per kilogramme of body weight than in a less reduced state of nutrition. The reasons for this will be discussed in the third lecture.

Generally speaking, the relative development of energy in an emaciating and an emaciated individual must be calculated in the same way as in an individual in a normal state of nutrition.

From 30-35 calories pro kg when resting in bed
From 32-35 calories pro kg when remaining quietly in doors
From 35-40 calories pro kg when performing a light amount of physical exercise (city dwellers)
From 40-45 calories pro kg when performing a moderate amount of physical exercise
From 45-60 calories pro kg when performing very severe labor

A diet that answers these requirements we call a "*Maintenance Diet*" ("*Erhaltungskost*"). If the diet remains below the above indicated caloric value, then we have a condition of *undernutrition,* and the inevitable result will be a more or less pronounced degree of emaciation.

V DISTRIBUTION OF LOSSES OVER PROTEIDS, FATS AND CARBOHYDRATES

In complete fasting the accumulated *carbohydrate* (glycogen) disappears very rapidly. The organism of an adult is capable of storing about 500 g of glycogen in the muscles and the liver. This is reserve material that is drawn upon as soon as the introduction of food ceases. At the expiration of 1½ to 2 days of fasting, it is probable that there are only traces of glycogen left stored in the different organs of the body.

The tendency of the organism to *spare its albumin content* as much as possible becomes clearly manifest during inanition. During the first four to five days of fasting an average of 18 to 20% of the developed calories are furnished from the disassimilation of albumins, later this figure falls to about 14 to 16%. Corresponding to this one finds a gradual reduction in the urinary nitrogen excretion in fasting subjects. The first two days show very great individual differences. The nitrogen excretion may fluctuate between 9 and 20 g. If the organs contained much glycogen at the time when fasting began, the nitrogen losses are reduced, owing to the albumin sparing power of the carbohydrates. If the individual was on an abundant albumin ration shortly prior to the period of fasting, high values for the nitrogen excretion are observed, (due to the high percentage of so-called circulating or better reserve, albumin).

The average excretion of	N in the urine	corresponding to dis assimilated albumin
on the 3d - 5th day of fasting	10 g	62.5 g
" 6th-10th " " "	8 "	50.0 "
" 11th-15th " " "	6 "	37.5 "

In women these figures average about 20% lower. In individuals, too, who had been in a condition of chronic undernutrition for some time prior to the period of complete fasting, the values are found to be somewhat lower than the above. This is due to the fact that attempts at sparing the albumin content of the body had already been inaugurated at an earlier period.

The organism is still more saving with the *substances composing the cell nuclei* and endeavors to preserve the integrity of the nuclear albumin even more so than that of the albumin contained in the blood plasma, the lymph and the protoplasm of the cells. In a fasting subject the individual cells emaciate and their protoplasm becomes narrower, while the nuclei remain intact and decrease very much less in volume in the early stages of inanition. It is only in advanced stages of inanition that the volume of the cell nuclei visibly shrinks; consequently the urine of a fasting subject contains only small quantities of the derivatives of the cell nucleus. In other words, the excretion of uric acid, a substance that together with the other purin bodies is derived from nuclein, becomes very slight, so that a fasting subject in the beginning passes from 0.3 to 0.4 g, later only from 0.2 to 0.3 g.

In a case of fasting that was studied with particular care by *Tigerstedt,* the development of caloric energy was distributed over the albumins and fats as follows:

Day of Fasting	Development of Calories		Source of Calories		Source of Calories in per cent.	
	Total	pro kg.	From Albumin	From Fat	From Albumin	From Fat
3	2024.1	31.2	339.4	1684.7	16.7	83.3
5	1970.8	31.2	286.1	1684.7	14.5	85.5

In chronic undernutrition the tendency also obtains to save the albumin content of the organism as much as possible, while at the same time sacrificing fats. It is due to this fact that individuals, who for long periods of time suffer from undernutrition and progressively emaciate, nevertheless maintain a considerable degree of efficiency during this period. The desire on the part of the organism to protect its albumin content, however, does not go so far that in undernutrition disintegration of albumin is completely avoided. This is only accomplished in subjects with very abundant adipose tissues (obesity). The amount of albumin sacrificed will depend essentially on the degree of undernutrition. A very common degree of undernutrition (found for instance in gastric disorders, in neurasthenia, in psychic depression, in fever) covers one-third of its caloric demands from the tissues of the body proper and only two-thirds from the food. I have found that in such cases fully 2 g more nitrogen were contained in the excreta than in the ingesta. Hence, an individual in this state of undernutrition would sacrifice in 100 days 200 g of nitrogen corresponding to 1350 g of dried albumin or 6 kg of flesh. These losses are considerably increased when, aside from the factor of undernutrition that causes a loss of albumin, a toxic factor incidentally injures the protoplasm (febrile infectious diseases, toxogenic and pyrogenic destruction of albumin).

It is frequently impossible to prevent chronic undernutrition, because the patients for one reason or another simply are incapable of ingesting or assimilating an amount of nutriment that would correspond to their maintenance requirements. In such instances the question arises, what character of food to administer by preference in order to prevent a highly injurious and justly feared loss of tissue albumin? The most natural step to take would be to increase the albumin ration and to arrange the diet in such a way that only the fats and carbohydrates remain deficient. A great number of albuminous prepared foods (proteids, albumins, caseins, albumoses, peptones) may be utilized for this purpose. I have found, however, that one does not accomplish very much, if on the one side the albumin ration is generally increased, while on the other, at the same time, undernutrition is allowed to persist.

For a few days, it is true, the loss of nitrogen is inhibited thereby, but very soon this loss is resumed and in many cases it even becomes increased.

Example: A young girl with gastric ulcer, weight 60 kg.; resting in bed. Calculated caloric requirement 1800 calories (60x30). Daily nutriment 2 liters of milk containing

66.2	g of albumin	à 4.1	calories	= 271	calories
62	g of fat	à 9.3	"	= 577	"
90	g of sugar	à 4.1	"	= 369	"
			Total	=1217	calories

Undernutrition—a deficit of about 33%. The daily nitrogen intake was 10.6 g; in the urine and feces were excreted an average of 12.4 g, signifying a daily deficit of 1.8 g.

There were now added to the diet of this patient 30 g of nutrose (à 13.6% nitrogen) containing 4.08 g nitrogen (25.5 g albumin, 104 calories). The albumin content of the diet therefore was considerably increased, that is, up to 91.7 g, and therewith attained a figure that could be considered completely normal. The caloric intake, however, was only increased to 1321 and consequently still represented a deficit of 479 calories, i. e., it was about one-fourth less than the calculated amount of the total caloric intake should have been (undernutrition with high albumin ration). The nitrogen intake and balance during the next 12 days were represented by the following figures:

Day	N-Intake	N-Output in Urine and Feces	N-Balance
1	14.68	12.9	+1.78
2	"	13.1	+1.58
3	"	13.9	+0.78
4	"	13.4	+1.28
5	"	15.0	—0.32
6	"	15.8	—1.12
7	"	15.1	—0.42
8	"	16.2	—1.52
9	"	16.0	—1.32
10	"	15.6	—0.92
11	"	16.4	—1.72
12	"	16.5	—1.82

The increase of the albumin ration therefore somewhat reduced the loss of nitrogen, but it did not permanently influence it. In a case of this character one would have been much more successful in protecting the albumin reserves of the body if instead of 30 g of nutrose 100 g of sugar had been added to the milk. This would not have been a difficult undertaking and would have increased the total caloric value of the diet by 410 calories, i. e., to 1627 calories. In this way the caloric deficit would have been almost wiped out and the property so characteristic of the carbohydrates to spare the albumins of the body would have caused the complete or almost complete inhibition of the nitrogen losses.

In cases suffering from severe loss of appetite or other difficulties that render it impossible to avoid undernutrition, I consider it advisable to arrange the diet in such a way that 25% of the energy supplied is furnished by albumen, the balance by non-nitrogenous food constituents. This proportion approximately corresponds to the distribution of nitrogenous and non-nitrogenous pabulum in milk. As soon as conditions become favorable for increasing the nutritive value of the diet and gradually compensating the caloric deficit, then it is easier to accomplish this goal and above all to spare and increase the albumen content of the organism by supplying non-nitrogenous food, specifically carbohydrate, than by adding albuminous food to the diet. Here an important law that has not as yet been sufficiently investigated and that is not generally appreciated becomes manifest, viz., that *with an increase of the food intake* the nitrogen balance determines towards equilibrium, even towards a positive balance, even when the total food intake remains one-half and even more below the calculated caloric requirement, scil., the maintenance diet. This becomes especially apparent if the small and gradual additions to the diet are supplied by carbohydrates. In such cases the paradox phenomenon is observed of an organism using up its own fat because the caloric intake is insufficient *while at the same time building up tissue albumen.* So great is the tendency of the cell to rehabilitate its albumen content even after days, weeks, and months of starvation. This also explains the important, well-established and frequently corroborated observation that convalescents regain their strength while they are still being underfed and are still consuming their own fat supply and losing weight. The increase in strength is a result of the energetic building up of tissue albumens.

VI THE EXCRETION OF ACETONE BODIES

A knowledge of the laws governing the excretion of acetone bodies during fasting is theoretically interesting and practically important. Sometimes acetonuria is a serious danger signal, at other times it may be simply one of the harmless consequences of faulty diet arrangement; in the latter case it would be very wrong to interpret the appearance of acetone bodies in the urine as evidence of threatening autointoxication. In order to understand the significance of acetonuria it becomes necessary to understand its relation to the quantity and the quality of the diet ingested.

The most important source of the acetone bodies are no doubt the lower fatty acids. These, provided they are not introduced pre-formed with the food, are derived within the organism from the catabolism of higher fatty acids. For reasons that are not yet fully understood the presence of carbohydrates is necessary, in order to promote the proper splitting and oxidation of the lower fatty acids. Possible that the co-operation of the carbohydrates in this process constitutes a " contact effect," possible that the fatty acids form a chemical combination with the carbohydrate molecule as a preliminary step to normal disassimilation. Whereas in process of normal degradation in the presence of carbohydrate, butyric acid forms in rapid succession oxybutyric, acetic acid and finally as end products carbon dioxide and water, this oxybutyric acid, in the *absence* of carbohydrate, forms diacetic acid and then acetone and water. The smaller the proportion of available carbohydrate the larger the proportion of fatty acids that is disassimilated by this route; and under these circumstances the destruction of the oxybutyric acid that is formed as an intermediary product also suffers, so that a certain amount of the latter acid also makes its appearance in the urine. The following formulae illustrate the normal and the pathological disassimilation of butyric acid:

1. CH_3
CH_3 = Butyric Acid
CH_2 (Common Mother Substance)
$COOH$

2. From butyric acid is generated by the addition of 1 atom of O
CH_3
$CHOH$ = β-oxybutyric Acid
CH_3
$COOH$

3. From here two paths are open, the one the normal and the other the pathological:

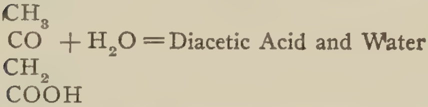
NORMAL

3 a. Still another atom of O is added while at the same time the chain is split, so that two molecules of acetic acid are formed.
CH_3
$COOH$
$\overline{\qquad\quad}\!\Rightarrow$ 2 molecules of Acetic Acid
CH_3
$COOH$

4 a. The readily combustible acetic acid is oxydized by the aid of 8 atoms of oxygen to 4 molecules of CO_2 and 4 molecules of H_2O.

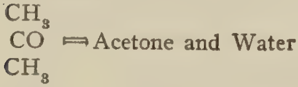
PATHOLOGIC

3 b. By the aid of one atom of O, 1 molecule of H_2O is split off—diacetic acid remaining.



$$\begin{array}{l} CH_3 \\ CO + H_2O = \text{Diacetic Acid and Water} \\ CH_2 \\ COOH \end{array}$$

4 b. The diacetic acid either maintains its integrity or is decomposed to



$$\begin{array}{l} CH_3 \\ CO = \text{Acetone and Water} \\ CH_3 \end{array}$$

It has been mentioned above that an individual completely fasting very rapidly consumes his glycogen reserves. Under these conditions carbohydrate becomes deficient in the liver cells where the degradation of the lower fatty acids chiefly, if not exclusively, occurs. In the absence of carbohydrate from the hepatic cells the decomposition of the lower fatty acids consequently occurs according to the pathological formulae delineated above. Inasmuch, however, as all the carbohydrate groups that are split off from the proteids still remain at the disposition of the organism in inanition, acetone bodies are excreted only in moderate quantities. Enough of acetone bodies is, however, excreted to produce a definite acetone and diacetic acid reaction in the urine by the second day of fasting. On the third and fourth days of fasting the intensity of this reaction increases considerably. On the second day one can demonstrate the presence of oxybutyric acid in the urine with methods, it is true, that are somewhat complicated.

Quantitative investigations show that the absolute quantity of acetone bodies excreted by a fasting subject fluctuates in each individual. The exact cause of these differences is not yet understood. The values fluctuate during the first four days of starvation as follows:

$$\left.\begin{array}{ll} & \text{From 0.5 –2.0 g of acetone + diacetic acid} \\ \text{and} & \text{from 2–13 g of oxybutyric acid} \end{array}\right\}\text{pro die}$$

It can readily be demonstrated that in inanition ketonuria is due to lack of carbohydrate. If 100 g of carbohydrate are given to a fasting individual, only 410 calories are being supplied with the food and the subject consequently remains in a condition of advanced undernutrition. This is demonstrated in the case report given below (Case A) where 100 g of carbohydrate nevertheless suffice to rapidly reduce the excretion of acetone or to completely suppress it. In Case B, below, the sugar was replaced by an equal quantity of pure readily soluble protalbumose made out of fish (trade-mark "Ribo"). This pabulum was completely absorbed and, although the quantity of proteid represented exactly the same caloric value, namely 410, as the carbohydrate administered in Case A, its administration exercised only a very slight effect upon the excretion of acetone bodies. This slight reduction in the excretion of acetone bodies is attributable to the fact that in process of degradation all proteids split off a certain amount of carbohydrate, varying according to the nature of the albumen. In interpreting the following tables it must be remembered that for the sake of clearness the total amounts of acetone bodies are calculated as oxybutyric acid.

	A			B	
Day of fasting	Oxybutyric Acid	Diet	Day of fasting	Oxybutyric Acid	Diet
1	4.8 g	1000 g water	1	3.6 g	1000 g water
2	4.8 g	1000 g "	2	3.6 g	1000 g "
3	6.1 g	1000 g "	3	4.7 g	1000 g "
4	3.1 g	1000 g " and 100 g sugar	4	5.7 g	1000 g " and 100 g albumose
5	3.1 g	the same	5	5.7 g	the same
6	0.4 g	the same	6	2.8 g	the same
7	Trace	the same	7	2.8 g	the same

The importance of carbohydrates in the normal dissimilation of fatty acids is so great that abundant quantities of the acetone bodies are formed and excreted even when a proteid-fat diet is administered that provides a sufficiently high caloric value to supply the caloric demands of the organism. One of my former assistants performed the following experiment on himself:

Day	Diet	Caloric value of the diet	Oxybutyric acid
1	200 g meat 200 g fat	2130	0.84 g
2	the same	2130	1.91 g
3	250 g meat 300 g fat	3228	8.73 g
4	0 g meat 250 g fat	2658	20.0 g
5	the same plus 150 g rice	3180	2.21 g

The weight of this individual and the amount of labor performed by him called for a caloric supply that was estimated as not to exceed 2300. Although the diet during the first two days incorporated nearly this number of calories and exceeded this figure on the two succeeding days, the excretion of oxybutyric acid nevertheless increased, owing to the lack of carbohydrate in the food; as soon as corbohydrate was added to the food on the fifth day, the excretion of oxybutyric acid promptly decreased.

Under these conditions it is not surprising to discover the development of acetonuria in a diabetic individual whose diet is suddenly changed from mixed feeding to feeding with a carbohydrate-free diet. This acetonuria in diabetes is frequently only a result of such a change in the diet and a phenomenon that is by no means specific of diabetes, but one that would occur in any healthy subject submitted to a similar change of diet. It is characteristic of diabetes, however, to see abnormally high degrees of acetonuria, and especially to see this large excretion of acetone bodies persist despite the addition of carbohydrate pabulum to the food. In the same sense the excretion of large quantities of acetone in patients suffering from high fever, severe acute gastric catarrh, gastric hemorrhage, uremia, etc., need not surprise us, nor should it be considered specific of any of these diseases; for some of these patients are incapable of ingesting more than a little meat broth and most of them eat nothing at all, so that one is dealing here with an acetonuria due to inanition or to lack of carbohydrates.

In chronic undernutrition even of long standing and when accompanied by advanced degrees of emaciation we relatively rarely encounter clearly demonstrable acetonuria. This is due to the fact that individuals taking small amounts of food as a rule eat a relatively large proportion of carbohydrate material. At first the amount of fat that is taken becomes reduced, then the proteid ingestion becomes smaller as the appetite sinks and the possibility of giving an adequate amount of food becomes less; not until the degree of undernutrition approaches actual abstinence from all food, fasting, does the intake of carbohydrates become so small that the fatty acids can no longer be disassimilated in a normal manner and acetone bodies are formed. When this point is reached, acetonuria constitutes a danger signal; and it is high time to employ all possible measures that can be directed towards preventing further undernutrition. Otherwise toxic conditions are very liable to develop that closely resemble diabetic coma. To this category belong so-called coma carcinomatosum, coma dyspepticum, etc. In all such cases, in which the introduction of food in sufficient quantities by mouth or by rectum becomes impossible, I advise infusion of a 10% solution of dextrose or levulose (1 liter a day). Such solutions introduced either subcutaneously or intravenously are very well borne.

VII. OTHER PROPERTIES OF THE URINE DURING INANITION.

Only two other properties of the urine in fasting may be discussed in this place; of these the former commands more theoretical, the latter more practical, interest.

(a) *The Urinary Ammonia* increases in fasting while the urea becomes correspondingly decreased. The absolute volume of the ammonia excretion also rises. This is attributable to the great production of acids that occurs in a fasting organism. In addition to sulphuric acid and phosphoric acid that are liberated from the proteids of the body, diacetic and oxybutyric acid also materially contribute to the increased acidulation. In any case of high acetonuria one can count upon finding high values for urinary ammonia and vice versa. It is essential, of course, that the urine examined should be fresh, as it will otherwise contain a certain amount of ammonia that is derived from the bacterial decomposition of urea after the urine was voided. Within the organism the acids combine with ammonia and by binding it in this way prevent its transformation into urea. The following example may illustrate the values that may be obtained:

Day of fasting	Total urinary nitrogen	Urinary ammonia	Ammonia expressed in per cent. of urinary nitrogen
1.	8.7 g	0.61 g	5.8 per cent.
2.	9.6 g	1.0 g	8.5 “ “
3.	9.6 g	1,3 g	11.1 “ “
4.	10.1 g	2.1 g	17.0 “ “

In chronic undernutrition an increase of the urinary ammonia is only found in the presence of acetonuria (see above).

(b) *The Urinary Chlorides* (Sodium Chloride) excretion becomes reduced during fasting to very low values. The depth of the excretion will depend principally upon the amount of sodium chloride that was ingested prior to the period of fasting. A certain time is required by the organism to get rid of surplus sodium chloride that may have accumulated. On the second, and, at the latest, by the third day of complete fasting, however, the urine rarely contains more than 1 g of sodium chloride in the total 24 hours' excretion, and after the fifth day of fasting rarely more than 0.6 g. This is diagnostically important. Every physician has, no doubt, heard patients declare falsely that for so and so long they had not eaten any food whatsoever. If in such cases the urine gives an abundant chloride precipitate with silver nitrate and if upon quantitative analysis several grams of sodium chloride are found in the 24 hours' quantity, then these findings may be considered valuable evidence towards disproving the veracity of the patient's statements.

In chronic undernutrition the excretion of sodium chloride also, of course, becomes reduced. No general rules can, however, be formulated in regard to the degree of this deficit, for everything will depend, not alone upon the quantity, but upon the quality of the food as well. Relatively the greatest decrease in the urinary chloride excretion in undernutrition is seen if, as is so frequently the case, milk alone is taken; for 2 liters of milk (representing about 1250 calories) contain only from 3 to 3.2 g of sodium chloride; and about this amount of chlorides are reclaimed from the urine in such individuals if this same diet is persisted in for some length of time.

VIII NUMERICAL DATA FOR CALCULATING THE DEGREE OF UNDERNUTRITION

The method of calculating the "Maintenance diet" (*Erhaltungskost*), has been described above (compare page 13). Whenever the total food intake, expressed in calories, remains below the calculated caloric requirement, then we speak of undernutrition.

The following numerical data are utilized to calculate the caloric value of the diet. Reference will be had to these figures in subsequent lectures:

1 g albumen	4.1 calories
1 g carbohydrate	4.1 "
1 g fat	9.3 "
1 g alcohol	7.0 "

We calculate the maintenance diet (provided there is no pronounced oedema), as follows (see also above):

		Example at 70 kg. body weight
about 30 cal. pro kg	patient in bed	2100 cal.
32 — 35 " "	patient confined to room	2240–2450 "
35 — 40 " "	light exercises (city dwellers)	2450–2800 "
40 — 45 " "	moderate physical exercise	2800–3150 "
45 — 60 " "	very heavy labor	3150–4200 "

In practice the calculation of the caloric value of the diet will not rest upon the caloric value of the basic ingredients of the food, i. e., its proteid, carbohydrate and fat, but rather upon the caloric value of the different articles of food; numerous tables giving this information have been published. For milk, cheese, butter, meat, eggs, fruit, bread and other bakery goods, raw vegetables, uncooked cereals and legumes these tables (aside, it is true, from certain sources of error), are useful and sufficiently reliable. In so far, however, as the amount of energy (caloric value) incorporated in prepared vegetables, soups, dishes made from flour, puddings, etc., is to a very great extent dependent upon their mode of preparation (consistency, sweetening, amount of fat) serious errors may creep in if too much reliance is placed upon average values as given for different articles in such tables. In all such cases the proportion of basic materials (proteids, carbohydrates, fats) must be known and included in the calculation. For instance, a cup of oatmeal gruel (250 ccm) prepared from:

20 g of oatmeal	furnishes 77 calories
(13.44% proteid 5.92% fat 67.00% carbohydrate)	
and 50 g butter	furnishes 405 calories
(1.2% proteid 86.7% fat)	
	Total, 482 calories

We speak of *a slight degree of undernutrition* if there remains a difference up to 20% between the maintenance diet (calculated caloric requirement) and the actually introduced food; of a *medium degree of undernutrition* if the deficit lies between 20 and 40%; of a *high degree* if the deficit lies between 40 and 60%; of a *severe degree of undernutrition* if the deficit exceeds 60%. Calculated for a man weighing 70 kg and resting in bed (see example above) this would give:

Maintenance Diet	2100 calories
Mild undernutrition	2100—1680 "
Medium "	1680—1260 "
High "	1280— 840 "
Severe "	less than 840 calories

IX COMPOSITION AND CALORIC VALUE OF IMPORTANT ARTICLES OF FOOD

100 Grammes	Albumen	Carbohydrates	Fat	Calories
Cow's milk (best quality)	3.4 g	4.8 g	3.6 g	67.1
Cow's milk (medium grade)	3.4 g	4.2 g	3.0 g	59.1
Sour milk	3.4 g	3.5 g	3.65 g	62.
Cream	Varying	Varying	10. g abt.	124
			15. g abt.	170
			20. g abt.	217
			25. g abt.	263
			30. g abt.	310
Butter (best grade)	1.2 g	....	86.7 g	811
Butter (medium grade)	1.0 g	0.1 g	82.8 g	774
Cheese (Gervais)	7.74 g	0.27g	49.2 g	489
Cheese (Dutch, Swiss, Camembert, Brie) average value	23.1 g	2.0 g	29.2 g	373
Hen's egg (without the shell) (1 egg about 45–50 g)	14.1 g	....	10.9 g	71
Caviar	26.5 g	....	14.3 g	241
Meat (lean) raw	20.9 g	....	1.6 g	103
Meat (moderately fat) raw	18.4g	....	5.2 g	136
Meat (fat) raw	17.0 g	....	10–25. g	163–300
Meat, boiled (lean)	30.0 g	....	4.0 g	160
Meat, boiled (fat)	25.0 g	....	10–35. g	193–425
Fish boiled (lean) average	20.0 g	....	1.0 g	91
Ham (lean)	26.4 g	....	3.6 g	141
Ham (moderately fat)	25.1 g	....	8.1 g	18
White bread	8.0 g	60.0 g	1.0 g	287
Black bread	6.2 g	52.5 g	....	242
Cakes, zwieback	7.0 g	73.3 g	4.6 g	374
Sugar	....	100. g	....	410
Cocoa (fat free)	20. g	40. g	31.6 g	537
Cocoa (inferior quality)	21.5 g	34.2 g	27.3 g	482
Bacon (smoked)	1–3. g	....	85–95. g	795–895
Oil	....	....	100. g	930
Rice (hulled)	6.5 g	77.5 g	0.9 g	354
Maize	9.45g	69.3 g	4.3 g	363
Oatmeal	10.7 g	58.4 g	4.9 g	329
Oatmeal flour	13.4 g	67.0 g	5.9 g	385
Potato	2.1 g	21.0 g	0.1 g	96
Peas (fresh)	6.3 g	12.0 g	0.5 g	80
Peas (dried)	23.1 g	52.7 g	1.8 g	328

PART II

FATTENING CURES

7/25

PART II

Fattening Cures

We are frequently confronted with the problem of improving the general nutrition of a patient with all the means at our disposal. Indications for this endeavor may lie in a considerable loss of weight and strength, in patients who formerly were in a normal state of nutrition, as a result of disease, overwork, inadequate feeding or an improper mode of life; many such individuals, it will be found, are incapable, unaided by medical advice and guidance, of regaining their former strength and good nutrition. In other cases faulty diet from childhood up must be blamed with having prevented certain individuals from really ever attaining the possible optimum of nutrition.

I DEFINITION OF THE STATE OF NUTRITION

Any attempt to gain information in regard to the state of nutrition of an individual must concern itself with two strictly to be distinguished inquiries, viz.: the condition of the protoplasm, i. e., of the muscular tissues, and the condition of the reserve materials, especially of the adipose tissues.

1. Muscular Tissues. The mass of the muscles, the condition and bulk of which can easily be ascertained, furnishes a measure of the content of the body in protoplasm. In estimating the latter, however, the large glandular organs and, above all, aside from the

muscles, the blood must also be considered. One can calculate that ordinarily the quantities of albumen in the musculature and in the blood run approximately parallel. An exception to this rule is found in certain diseases, as special diseases of the blood-forming organs, neuropathic and myopathic atrophies, etc. An individual with a scanty musculature is always also suffering from atrophy of the blood; here an examination of the blood need not necessarily reveal any deviations from the normal in its *composition,* but the *mass* of the blood is reduced. Some authors designate this condition of the blood as *oligaemia vera,* others probably more correctly, as an atrophy of the total blood. In absolute fasting and in chronic undernutrition valuable proofs for this contention are found, the mass of the blood decreasing proportionately to the loss of muscle tissue, whereas pathological changes in the composition of the blood do not appear until later. In chronic pulmonary tuberculosis one also sometimes witness a similar sequence of events, viz.: high degrees of emaciation, great pallor of the skin and of the mucous membranes, but nevertheless almost normal blood findings. Whenever the blood suffers more than the musculature then special factors that inhibit blood formation or that accelerate blood destruction must be operative. In all such cases the blood composition will always be found abnormal (oligocythemia, low percentage of hemoglobin, thinning of plasma, etc.).

No optimum can be formulated for muscular development or for muscle bulk. We should be content to attain the greatest development of which each individual is capable. Definite limits are set to the development of the musculature by individual peculiarities and often hereditary factors, that consequently may be called endogenous. Not every individual can be developed into an athlete even though every effort may be made in this direction from childhood up. Here and there special conditions may obtain that render it inadvisable to aspire to the highest individually possible muscular development, as, for instance, cardiac disorders, certain forms of paralysis, deformities, etc. As a rule, however, that degree of muscular development that can be obtained without detriment to the organism in general or to special organs should be striven for. Essentially the same considerations are valid in the case of the blood; true, one is here always haunted by the fear of "plethora"; and, as a matter of fact, there seem to be conditions in which —aside from hydraemia—the mass of the blood becomes increased beyond a point that is of value to the organism and that exceeds the average degree to which the bloodvessels should be filled. Why this condition, that has been designated as "hypertrophy of the blood," in contradistinction to the "atrophy of the blood," mentioned above, should be particularly common in chronic respiratory and circulatory disorders, often associated with obesity, we do not know. Modern investigation has adopted an attitude of negation towards the old, clinically by no means unimportant conception of "plethora," that is hardly justified. Finally there remain to be mentioned certain rare and etiologically also obscure cases of polyglobulemia (erythrocytosis).

Aside from these exceptions the axiom can be maintained that the two main albumen carriers of the body, the muscles and the blood, develop, increase and decrease in a parallel manner.

2. Adipose Tissues. Whereas in the case of muscular tissue one cannot very well formulate an optimum of nutrition, one can readily do so in the case of the adipose tissue of the body. Here one is justified in speaking of a "medium condition of nutrition" and in declaring this to be the optimum. This optimum, however, has fairly wide boundaries. Expressed numerically it presupposes a fat content of from 18 to 22% of the body weight in an adult man, of from 25 to 28% in an adult woman. The layman who judges exclusively from external appearances will never hesitate to designate an individual as thin, normally developed, or fat. As a matter of fact, however, the situation is very complicated. In the first place it is always important to determine the relation between the musculature and the adipose tissue and to include it in the calculation. In order to do this properly one must have medical training and education, and there are other questions besides that a physician alone can decide. He knows, for instance, that under certain pathological conditions an adipose

development that exceeds the average is desirable as, for instance, in tuberculosis and diabetic subjects. He knows that in numerous neurasthenics, even if their muscular development is very great, all attempts at a cure become abortive unless one succeeds in increasing their reserve material; this applies particularly to sufferers from gastric and intestinal neurasthenia. This peculiar phenomenon is difficult to understand, but it is constantly being corroborated by practical experience. In other morbid conditions on the other hand it may appear desirable to reduce the fat of the body to a point that lies below the "medium state of nutrition." To this category belong numerous disorders of the respiratory and circulatory apparatus, as well as certain neurogenous, myogenous and arthrogenous disorders of the organs of locomotion, particularly about the lower extremities.

It will be seen, therefore, that the question whether or not an individual is in the optimum of development as far as his adipose tissues are concerned can only be solved if, aside from external appearances, the relation between the musculature and the adipose tissue, as well as a series of individual factors, are all considered. In most cases the matter is quite clear and very apparent. In others again it may become necessary to carefully weigh all the circumstances of the case before active modification of existing conditions is undertaken by the physician.

II CONDITIONS PRELIMINARY TO A FATTENING CURE

The determination of the "Maintenance Diet" (*Erhaltungskost*) is a necessary preliminary to any endeavor that may be made towards increasing a reduced state of nutrition. In a previous paragraph (see page 13) the method of calculating this factor has already been described. It is clear from what has been said above that the maintenance diet fluctuates within broad limits according to the condition of the body and according to the demands made upon its musculature. For a definite body weight (70 kg) we calculate variations lying between 2100 and 4200 calories. There is, therefore, no diet that can be called a "fattening diet" that is based upon any definite standards—unless we exclude certain extreme conditions that are rarely seen. A certain diet may be very fattening for one individual, while it may induce undernutrition with considerable loss of weight in another individual. The following example may illustrate this:

2 l milk	1340 calories
200 g white bread	574 "
100 g butter	811 "
6 eggs (300g)	213 "
	Total, 2938 calories

An individual of an average bulk weighing 80 kg and performing an ordinary amount of labor could not remain in nutritive equilibrium on a diet of this kind, because his caloric demands lie between 3200 and 3600 calories a day. In other words, on such a diet this individual would progress to a condition of undernutrition of a mild degree.

Such a man would without doubt lose weight (approximately from 300 to 400 grammes a week) and grow weaker.

For a reduced tuberculous individual weighing 60 kg and resting in bed the same food would be an excellent fattening diet, for the demands of such an individual would be 1800 calories, and the surplus pabulum of about 1100 calories would enable this individual to gain about 1 kg a week.

These examples seem so self-evident that it might be considered superfluous to mention them at all. Matters, however, are never quite so clear as delineated above, and I consider it necessary to advise a careful estimation of the maintenance diet, because I continually encounter instances in practice where serious errors are committed simply because these simple calculations are neglected. Under such conditions both the patient and the physician object that all attempts at fattening remain futile; the patient is considered unsuitable for a fattening cure and time, trouble and money are considered wasted with further attempts in this direction.

If in such cases the diet that is being used as a fattening diet is carefully calculated, one will find, however, almost without exception, that no true conception was arrived at in regard to the real demands of this individul for food in regard to the nutritive value of the so-called "fattening diet," and that the two, i. e., demand and supply, were not arranged in proper proportion.

It is, of course, also very important to know what increase in weight can be justly expected in a fattening cure. I have been collecting material of this kind for several years and can indicate the following values as representing an average:

Daily surplus of food (or "fattening addition")	Weekly increase in weight
500 – 800 Cal.	600 – 1000 g
800 – 1200 "	800 – 1200 g
1200 – 1800 "	1200 – 2000 g

I offer as a definition of a surplus of food or a fattening addition the sum of the nutritive units (calories) administered in excess of the calculated nutritive demands (Maintenance Diet) of the individual.

III INCREASE OF MUSCULAR AND ADIPOSE TISSUE

I have already mentioned that the amount of muscle tissue and the amount of adipose tissue of the body must be estimated from two altogether different standards. The two are to a very great extent independent of one another. We learn this from daily experience; for we frequently encounter very muscular men who have very little fat, and, on the other hand, obese subjects with a very scanty muscular development. It is well also to recall the fact mentioned in a previous lecture that the albumen content of the body may become increased while at the same time the adipose tissue becomes decreased and the weight reduced (see page 19). The increase of the muscular and of the fat layers must, therefore, be discussed separately.

1. INCREASE OF MUSCULAR TISSUE. Whenever an individual is overnourished—immaterial whether the caloric surplus is furnished by proteids, fats or carbohydrates—it will be found upon investigation of the nitrogen balance that a certain amount of nitrogen is retained in the body. Many hundreds of exact studies of this kind that, of course, cannot be carried out in general practice, but that offer no difficulties in a clinic or a laboratory, have been made. Whereas the fact is established that under the influence of overfeeding nitrogen retention occurs, the significance of

this factor has not yet been cleared up and there is still much discussion on this subject. When the science of metabolism was still in its infancy an explanation was promptly forthcoming, for the investigators of that time simply calculated the retained nitrogen as albumen and later as muscle tissue according to the following figures:

1 g of retained N = 6.25 g of albumen = 29.4 g of muscle tissue

Doubts in regard to the validity of this explanation were expressed early by the father of the science of metabolism, *C. von Voit,* for he recognized the uncertainty of the explanation that the nitrogen retention could be interpreted directly as a gain in muscular tissue. The same investigator also noticed how easily later on, when the ingestion of food was reduced, this retained nitrogen was again eliminated. This phenomenon was in marked opposition to the ordinary observation in cases of normally fed or undernourished individuals in whom upon the reduction of the proteid ration the organism very obstinately retained its nitrogen (compare first lecture). *Voit* constructed the conception of labile so-called "*circulating albumen*" in contradistinction to a more stable "*organ albumen.*" Before this time proof had been furnished that any albumen acquired by overfeeding was not found present in the circulating fluids of the body. Later (in 1893) I formulated the hypothesis that the retention of nitrogen actually represented a building up of albumen, but that this albumen was stored in the cells themselves as *"so-called reserve albumen"* similarly to glycogen and fat that can also be deposited in the cells as reserve material. I expressed the opinion at the time that the albuminous reserve material contained in the cells could impossibly possess the viability and the value of protoplasm proper and could not participate like the latter in the transformation of energy. For this reason I coined the word "dead cell inclosures"; this expression has been thoroughly misinterpreted by other writers. I confess that it was not very happily chosen, because one can hardly designate any constituent of the living body as "dead." The sense, however, in which the word was employed should have been altogether clear, and later investigations, as I will have occasions to show, endorsed the views expressed by me at the time. Now-a-days there is no doubt whatsoever that the nitrogen retention seen in overfeeding really indicates a true addition to the albumen stores of the body and that the cells are the depot in which this excess albumen is stored. A small portion of this retained nitrogen is possibly built up no further than to the stage of certain precursors of the albumen molecules that, like the first disintegration products of the albumen molecule, may be designated as peptids and polypeptids.

The amount of nitrogen that can be retained in the body by overfeeding varies. The quantity of retained nitrogen is dependent—leaving aside certain important factors that are to be discussed later—chiefly upon the amount of albumen introduced, the kind of albuminous food offered and the degree of the caloric surplus. If the diet contains much albumen, and especially if the albumen it incorporates consists chiefly of casein (or nutrose, plasmon or nucleo proteids) then the increase of body albumen becomes particularly great. If there is a preponderance of meat, and especially of eggs and vegetable albumens among the albumens of the diet, then the building up of body albumen is smaller. When all the contributing factors are favorable one may count upon a daily retention of from 5 to 6 gn (albumen 31 to 38 g). In ordinary fattening cures with a moderate albumen ration (about 100 to 120 g daily) and a moderate caloric surplus (of 30 to 40% above the calculated amount of the maintenance diet) one may expect to find a daily retention of nitrogen of from 1 to 3 g; if the caloric surplus is from 40 to 60% above the maintenance diet, one may expect a retention of from 2 to 6 g of nitrogen *per diem.* Calculated for each day this appears a very small sum; if this factor is repeated for long periods of time, however, very considerable additions result.

In an experiment carried out with painful accuracy by my former assistant, *L. Mayer,* 383 g. of nitrogen were retained during a period of 62 days. Using the ordinary method of calculation, one finds that from this amount of nitrogen can be calculated the retention of 2394 g of dry albumen or of 11.26 kg of meat (cellular substance). The individual upon whom this test was made was, prior to the beginning of the experiment, in a fairly good state of nutrition and the attempt to fatten him was made only for the sake of the experiment and with his full consent. The body weight increased during the experiment from 56.0 kg to 69.5 kg.

Two important observations have been made in regard to nitrogen that has accumulated in the body in this way by overfeeding. The first of these observations is of constant occurrence; the second one, requiring for its verification an enormous amount of tedious work, has been verified only once (namely, in the above quoted experiment of *L. Mayer*).

1. The nitrogen bodies (or as we may say with equal safety), the albumen that has been accumulated in this way, does not show a great tendency to remain in the body; in other words, it does not "stick" very hard. As soon as the albumen ration or the amount of nitrogen-free food is reduced a large proportion of the previously accumulated nitrogen reappears in the urine as urea. The individual for instance upon whom *Mayer* experimented eliminated 24 g of nitrogen in the urine during the 21 days following the experiment, although the diet still contained the considerable sum of from 100 to 118 g of albumen and although pabulum representing in its totality an average value of 43 calories pro kilo was being introduced; in other words, really more than would correspond to the calculated caloric requirement of an individual remaining in one room and performing a very small amount of physical labor. Newer experiments therefore completely corroborate the find-

ings of *C von Voit's* animal experiments (see above). It would be important to determine by a prolongation of these experiments (for instance by prolonging the so-called after-period that sets in as soon as the fattening period is terminated) how long the accumulated albumen really remains in the body. So far no investigation has been continued for a sufficiently long time to enable us to arrive at definite conclusions in regard to this point. This much remains established, however, that a large portion, possibly all of the albumen that is accumulated in the organism of an individual who was previously well nourished, does not possess the same stability as the albumen that was originally present in the body. It is probable that the physiologic dignity of the latter albumen is not as great as that of the former.

2. We know that the development of energy that the organism puts forward is a function of the albuminous protoplasm of its cells. If we were able by overnutrition to increase the quantity of the genuine energy-consuming protoplasm of the cells, we should be able to increase the oxygen consumption of the body proportionately. In the individual utilized in the experiment of *Mayer,* the consumption of oxygen per minute, however, was only increased from 222.4 to 226.1 ccm at a body weight of from 56 to 57 kg, to 234.1 ccm at a body weight of from 68 to 69 kg.

This small increase in the oxygen consumption is readily explained by the increase in weight, and this increase is strikingly small. Calculated per kilo of weight, the consumption of oxygen actually decreased; for in the beginning it was 3.987 ccm pro kg a minute; at the end 3.373 ccm. One is therefore justified in formulating the conclusion, a careful justification of which need not be attempted in this place, that the accumulated albumen is not concerned in the process of internal respiration, but possesses a different dignity from the protoplasm that was originally present in the body.

The conclusions, therefore, that can be drawn from the first and the second observations coincide, and it appears that my original hypothesis, that bestowed upon the albumen that was accumulated by overnutrition merely the significance of passive reserve material and not the importance of active protoplasm, is hereby strengthened and corroborated. From all that has been mentioned above, therefore, *we are not justified in concluding that by overfeeding alone —without the co-operation of other factors—a material increase of "flesh" (genuine breathing protoplasm) can be forced.*

The real "accumulation of flesh" seems to be dependent on altogether different factors and seems to presuppose a specific predisposition on the part of the organism to accumulate flesh. We find a ready tendency to the increase of flesh in

1. the growing organism.
2. during convalescence, that is, after a certain amount of albumen has been previously sacrificed by disease or by inadequate nutrition.

3. in muscles (and glands) that are stimulated to greater activity by gradually increasing and systematically carried out exercise of their function (labor hypertrophy).

Relative to points 1 and 3 there are exact metabolic investigations to prove their correctness. It was found that with an increasing accumulation of nitrogen the consumption of oxygen increased, a fact that could not be determined in the fattening experiment of *L. Mayer.* Relative to point 2, a number of experiments have also been performed, but they do not suffice to give us a clear conception in regard to the intensity and extent of the flesh accumulation and the increase in the consumption of oxygen that takes place. I am just about to undertake a study of this character myself.

If one or the other of the three above mentioned factors is operative the tendency to an accumulation of flesh becomes extraordinarily marked; in fact, this tendency becomes so irresistible that the accumulation of flesh may even proceed under unfavorable conditions of nutrition. I have already mentioned (see page 19) that in individuals convalescing from certain diseases albumen may accumulate, even though these individuals remain undernourished. The growth of children also may become retarded by undernutrition, but not completely arrested. They do not gain in weight and remain horribly emaciated, but the growth of the body nevertheless indicates with certainty that albumen is being retained and protoplasm stored. If anyone still maintains that by overnutrition muscle, blood and glandular tissue can be built up, then he should become convinced of the futility of this argument by the daily experience that by overnutrition alone an individual can be made fat but can never be made an athlete.

We must further learn to realize that while fattening cures may also be of benefit for the development of the body musculature their action is altogether indirect. The first step is an increase in the reserve material (glycogen, fat, albumen inclosures in the cells) that the organism stores. *The musculature, provided favorable factors co-operate, can then draw from these accumulated reserves the material that it requires to promote actual growth of muscle tissue.* Up to a certain degree it lies in our power to create suitable factors that can become operative toward producing this result. In other words we must combine, as far as that is possible, muscular exercise, carefully in the beginning and gradually increasing, with overnutrition.

Inasmuch as I am expressing such unfavorable judgment in regard to the immediate effect of overfeeding upon the increase of protoplasm, the question might very justly be asked: why then undertake a fattening course at all, if the fat that accumulates is of very little value; why not from the beginning exercise and train?

To this query one must reply that objections of the kind just formulated go much too far and do not harmonize with practical experience. Even under unfavorable conditions of nutrition the protoplasm in an emergency can develop and grow, although not very efficiently. The protoplasm of the body can only develop in an efficient manner and reach the optimum that it was intended to reach by heredity and by the individual laws of growth if a certain surplus of nutrition is at its disposal. Hence it is beneficial, not only as far as the accumulation of adipose tissue but also as far as the development of the cells is concerned, to put forward every endeavor to place a poorly nourished organism into conditions of better nutrition. A muscle that is starving does not become strong, or at all events the strengthening process remains slow and does not reach its maximum.

Certain factors other than the lack of reserve material can prevent this development, as, for instance, the co-operation of certain toxic agencies. We know that numerous pathogenic micro-organisms and malign tumors are capable of producing toxins that act as so-called "protoplasmic poisons," and we also know of a large number of other organic and inorganic poisons that possess the same property. So long as protoplasmic poisons of this kind continue to circulate in the organism, the development and growth of the cells is seriously impaired; and all known empiric and biologic laws would be violated if one were to attempt by forced feeding or by training to increase the flesh of a typhoid patient or of a phthisical individual with fever.

The history of therapeutics unfortunately reveals many errors of this character, all amounting to this, that the factors that are really important in the construction and the destruction of the tissues are underestimated, while at the same time, the futile attempt is made to influence the growth of the tissues by introducing substances into the organism contained in these tissues. On this basis iron was prescribed in chlorosis, because an impoverishment of the organism in iron was assumed and considered to be the cause of the deficient formation of haemoglobin. By the merest chance the correct therapy was discovered in this particular instance, although the underlying idea was wrong. Iron must be considered to be merely an irritant of the bone marrow, and numerous other substances that have no relation whatsoever to the haemoglobin molecule act in precisely the same way, for instance, arsenic. Manganese also, as well as very minute quantities of copper and certain other nonmedicinal measures exercise the same effect upon the bone marrow in chlorosis.

In the same sense calcium salts were administered in rachitis and in osteomalacia, of course without success; for there is never a lack of calcium in the food, especially not if cow's milk, which is very rich in calcium, is taken. The deficient assimilation of calcium or in another sense the decalcification of the organism is in reality dependent upon a disordered function of the bone cells, and an excess of calcium does not correct this perversion. The idea of strengthening a neurasthenic individual by the administration of lecithin—"the specific pabulum of the nervous substance," as one advertisement claims—is equally irrational. An ordinary mixed diet, especially if it contains 1 to 2 eggs, incorporates more assimilable lecithin than is required by the whole organism. The same criticism can be formulated in regard to the use of so-called physiologic salts that are being sold in manifold combinations and under a great variety of trade-marks.

All these and many other similar prescriptions are altogether lacking in true tissue-building power. The same applies to so-called fattening foods. The regulatory energy of the cells that is expended in the direction of promoting their growth always enters into the equation. One is capable by undernutrition to paralyze this power, but one is capable only to a very moderate degree of increasing it by overnutrition; in fact, it is quite probable that this power cannot be increased above the point that it would attain when the organism is receiving its proper "maintenance diet."

2. INCREASE OF ADIPOSE TISSUE. From all that has been said it is clear that to increase the adipose tissue is much simpler and much more certain than to increase the protoplasm. *One can force an accumulation of fat by increasing the amount of food that is introduced and that is assimilated above the standard of the maintenance diet.* In fact, one is able to predict with almost mathematical accuracy how much

fat can be deposited in this way. If we add a definite surplus to the maintenance diet (fattening addition), then a small proportion of the superfluous calories is utilized in the process of digestion and absorption (an average of about 8%); a small part leaves the body without having been utilized at all (unabsorbed material, losses in the feces averaging about 4% of the caloric surplus). Of the large remainder a small amount, averaging at the utmost 10% of the caloric surplus, is stored in the cells in the form of reserve albumen. All the rest is deposited as nitrogen-free material. It is customary to calculate this latter factor as fat, although one must remember that a portion of this surplus is also stored as glycogen. The latter portion, however, is very small, and at the expiration of a few days reaches a maximum that cannot be exceeded, so that from then on the calculation of the stored material as fat corresponds to actual conditions.

As an example the following fattening experiment, that *B. Krug* performed eighteen years ago under my direction, may be analyzed:

	Duration	Body weight at		Diet pro die				Calories pro die and kilo	N-Accumulation pro die
		Beginning	End	N	Fat	K H	Calories		
(Maintenance diet) Fore period	6 days	59.0	59.4	14.8	151	192	2575	43.5	-0.402g
Fattening period	15days	59.4	62.5	15.4	227	425	4285	70.0	+3.30g

The surplus food over and above the maintenance diet amounted to 1710 calories (4285–2575) daily.

Deduction for labor of digestion	8%	137 calories
Deduction for losses in the feces	4%	68 “
Addition as albumen (20.69 only) (1 g organ albumen 5.5 calories)	6.6%	113 “
Balance for addition of fat	89.4%	1392 “

As 9.3 calories correspond to 1g. of fat about 150 g of fat were deposited each day.

Calculations of this kind can, of course, only be performed in an exact manner if analyses of all the articles of food and of the excreta are made at the same time. In general one must be content with estimates that are sufficiently accurate, however, in practical work.

In determining simply the increase in weight other values must, of course, be introduced than in determining the deposit of albumen or of fat; for the bulk of the body is increased not only by dry substances but also by water and by inorganic constituents. In a previous paragraph (page 38) I have given certain figures relative to the latter point that I have gained from an experience extending over many years.

IV FATTENING FOODS

From what has been said it is quite clear that all articles of food that incorporate caloric values that become available for the organism may also be considered articles that can be used for fattening purposes. The *availability* of the caloric content for the organism is, of course, a fundamental condition and it is only this property that characterizes any article that is eaten as a food. Carbon and hydrogen, for instance, have an enormous caloric value. They are incombustible, however, in the organism, and hence constitute no source of energy for the latter and are not foods. Another condition that the introduced material should fulfill is that it must be non-toxic and in no wise injurious to health. This point must always be considered in judging the value of alcohol as a food (see below).

Fundamentally it is immaterial which one of the main articles of food is employed for fattening, immaterial whether we use proteids, fats or carbohydrates. Here the practical rather than the theoretical point of view must be considered, and such practical considerations may lead us in different cases to employ different articles of food; for instance, one would never attempt to increase the weight of a diabetic with carbohydrates, of a gouty individual with meat, of an icteric individual with fat. In each case an individual study of the metabolism and of the capacity of the digestive apparatus must be carefully made; otherwise failure is assured from the beginning.

Aside from these peculiarities the different articles of food can be employed vicariously, according to the well-known law of isodynamics, in so far as their fattening value is concerned. That is, such quantities of the different articles are equivalent that introduce into the organism an equal quantity of available energy.

As 1 g albumen	introduces	4·1	calories
1 g carbohydrate	"	4·1	"
1 g fat	"	9·3	"
1 g alcohol	"	7·0	"

The following proportions of these articles may be considered equivalent, viz:

100 g fat	930 calories
227 g albumen	
227 g carbohydrate	
133 g alcohol	

As long, however, as one is confronted with the task of accomplishing the greatest possible good with the least possible inconvenience to the patient, certain practical standpoints enter into the equation, and it will then be found in practice that the different articles of food are by no means equivalent. It is necessary, therefore, to briefly discuss the different articles of food.

1. PROTEIDS. Proteïds do not possess great fat-

tening value and no other class of foods requires so large an expenditure of energy on the part of the digestive apparatus in proportion to the calories placed at the disposal of the organism. On a mixed diet, as we have seen, about 8% of the energy incorporated in the food must be charged to this account. In the case of the proteids, however, as much as 12 to 15% must be charged to the labor of digestion itself. In addition, proteids act as a bellows upon the oxydative processes (so-called dynamic action of proteids, *M. Rubner*). A proteid diet, therefore, stimulates a useless consumption of a part of the total energy that it is intended to force the organism to store. This peculiar action is very considerable and may lead to a destruction of almost half of the energy contained in the surplus diet, provided the latter is administered exclusively in the form of albuminous pabulum.

An individual fed on an ordinary maintenance diet ingests daily an average of 100 g of proteids. Practically it is impossible to increase this quantity above 150 g, and we thereby gain an increase of only

$$50 \times 4.1 = 205 \text{ calories.}$$

Even 150 g of albumen given daily are frequently too much, and are rarely tolerated for any length of time, even by individuals who are originally heavy meat eaters. Subjects who are undergoing an artificial fattening cure are usually afflicted with a weak appetite. Large quantities of albumen still further damage the appetite, probably because they impose more labor upon the gastric function than any other article of food and because the stimulus of appetite emanates more from the stomach than from the intestine. If large quantities of albumen, therefore, are given, it usually becomes a difficult matter to incorporate in the diet the desired increased quantities of other articles of food, and it is precisely these other articles of diet that must be utilized to increase the total caloric intake. I can hardly advocate the plan of administering concentrated albumen preparations with the object in view of increasing the proteid ration without giving too large volumes of albuminous food. I am particularly averse to the use of insoluble albumen powders (Nutrose, Plasmon, Roborat, Tropon, etc.). Whenever I have been induced to utilize these preparations, I have always found them to be rather a detriment than an advantage in any attempt to increase the total intake of caloric values. A somewhat more favorable verdict can be expressed in regard to soluble albumen preparations. While the total calories that one can introduce with them is not very great (often corresponding only to 20 g of dried albumen or 84 calories), they are nevertheless useful and convenient in practice, because in such small quantities they act as a distinct stimulus to the appetite. Just why this should be so remains for the present undetermined. There is a great variety of these soluble albumen preparations. My personal experience is essentially limited to Somatose (an albumose made out of meat) and to Ribo (a readily soluble albumose made out of fish, but yet not on the market). The latter product is tolerated better than Somatose and it does not possess the disagreeable property of producing diarrhoea. It is possible to increase the daily intake of Ribo up to 60 or 80 grammes, and this particular albumen preparation is almost completely absorbed.

The optimum of the albumen intake in fattening cures lies in my judgment between 100 to 120 g. Such quantities are usually incorporated anyhow in the diet that one uses in fattening cures (meat, fish, eggs, milk, etc.); if not, this amount can easily be supplied by one of the preparations mentioned above.

I herewith submit a small table giving the quantities of several articles of food containing 100 g of albumen:

Beef (weighed raw)	480-550 g
Veal, Chicken (weighed raw)	500-550 g
Eggs (without the shell)	900 g
Fish (weighed raw)	500-600 g
Cow's milk	3-3 1-2 l
Cream cheese	400-450 g
Cottage cheese	250-300 g
Sanatogen	105 g
Tropon	110 g
Plasmon	125 g
Somatose	120 g
Ribo	105 g
Puro	300 g

2. CARBOHYDRATES. Carbohydrates have always

enjoyed great popularity as a means of fattening. Most of them can be prepared in a very palatable form and there is an almost unlimited variety. In the preparation of this class of foods, the tastes of the patient, as well as the digestive powers of the stomach and intestines, can to a wide extent be considered. Aside from diabetes mellitus and certain forms of fermentative dyspepsia, there is no disorder in which these foods cannot be used extensively in order to produce a gain of weight. Nevertheless one should expect occasionally to encounter difficulties when they are given in large quantities, for the power of the digestive apparatus to absorb carbohydrates varies greatly in different people. Individual and geographic peculiarities and habits have to be considered. In Southern Germany and in Austria-Hungary and in most of the Romanic countries almost twice as much carbohydrate is eaten by the natives as in Northern Germany, Scandinavia and large parts of Russia. Among the former one can figure on a daily adult consumption of 400 g of carbohydrate, among the latter of 200 to 300 g. The consumption of fat is exactly the reverse.

One important element must be considered in the administration of a one-sided carbohydrate diet that often hinders attempts to produce fattening by this means, namely, that most carbohydrates, if prepared in a palatable form, become so bulky that their volume is out of all proportion to the caloric value that they incorporate. It is true that there are certain foods made of flour that incorporate a high percentage of carbohydrate, as for instance, bread and other bakery goods (averaging 60 to 70% carbohydrate), and rice and macaroni with an average of from 30 to 40% carbohydrate (in a properly prepared form). Unfortunately these highly concentrated foods made from flour are not of great value in fattening cures, because they are too filling. In fattening cures the available carbohydrate foods must be calculated to contain on an average not more than 20% carbohydrate (either in the form of flour or in the form of sugar). For this calculation of averages the following articles of food have been considered: milk, soup, gruel, purées, dishes made from flour, desserts, fruit, cheese, fruit sauces and fruit.

The average voluntary ingestion of carbohydrate in an individual that is to be fattened probably does not exceed 180 g. If the carbohydrate foods are properly distributed over the different meals, and if the proper amount of carbohydrate food is selected, this amount can without great difficulty be increased to about 320 g during the course of the fattening treatment. This signifies an increase of 140 g, containing 570 calories, certainly a considerable sum; 320 g carbohydrate, however, require a volume of 1600 ccm of ingesta. This is not too much, for there are included in this volume the greater part of the fats that are to be taken. It is difficult from the standpoint of the cook to include more than a small portion of the albuminous foods in this volume. To administer them a further increase of the volume of the food becomes necessary.

If now one attempts to give still more carbohydrate then the volume of the food increases so much that difficulties in its administration arise. Under direct medical supervision and under the régime of a sanatorium, etc., it is possible, of course, to give even greater quantities of carbohydrate. *Kuszmaul,* for instance, was in the habit of giving enormous quantities of carbohydrate (in the form of soup, gruels, mushes, etc.) in his fattening cures; in fact he gave as much as 400 to 500 g of amylaceous foods. As soon, however, as the patients were dismissed from the institution and were no longer forced to eat these large quantities of carbohydrate food, they lost the accumulated flesh because they reduced their food intake. I have often witnessed a disappearance of fat accumulated in this way by one-sided carbohydrate feeding within a short time. The volumes of food are too great. I should consequently advise not to exceed 300 to 320 g of carbohydrate in general. In this way an introduction of from 1230 to 1310 calories is always assured. Only in those cases in which saccharine fruit juices can be employed would I advise as practical the attempt to introduce more carbohydrate. I am thinking in particular of unfermented grape juice, a bottle of which is generally willingly taken during the day. This beverage is very refreshing and nourishing and at the same time possesses mild laxative properties, an effect that is very desirable in many patients. The pasteurized grape juice from Istria (*A. Cerovaz* in Bruck a. d. Mur) contains 18% of inverted sugar and incorporates in a bottle of 750 ccm not less than 550 calories. I have frequently prescribed a bottle of this preparation as the sole fattening addition to the diet with good results in cases in which it was otherwise impossible to increase the food above the maintenance diet. A result of this kind is not surprising when we consider that the 550 calories of surplus furnished in this way imply a daily fat deposit of about 60 g.

In addition to the ordinary carbohydrate foods that are available in any ordinary bill-of-fare, there are a large number of other preparations containing abundant carbohydrate on the market. An old-established adjuvant is malt extract. One can easily add 50 g of *Hoff's* Malt Extract to the milk each day without spoiling its taste. One of the cereal preparations called *Kufeke's* Children's Flour (100 g = 370 calories) that is so popular in pediatric practice may become a welcome addition to the diet in difficult cases, particularly in the beginning of a fattening cure. In general too little use is made of these artificial carbohydrate foods. In America a much greater variety of these products is available than in Germany. In the United States, as well as in many regions of England, it is customary to eat a cereal for breakfast, usually with thick, sweet cream. Such a mixture constitutes a very nourishing and readily digestible food. I mention in particular porridge made from oatmeal, then a thick gruel made from hominy (a mais preparation) and also Grape Nuts, a preparation that has recently become very popular. The latter is made of slightly roasted wheat. I have learned to appreciate this preparation as a particularly useful adjuvant in fattening cures. A very practical and nutritious method of preparing it is the following: About 40 g of Grape Nuts are heated with a little water for 15 minutes in a water bath, then thoroughly mixed with 40 g of butter and served with 60 cc of thick (40 per cent.) cream. This very palatable dish incorporates in a small volume not less than 650 to 670 calories. All these preparations are useful, less on account of their inherent nutritive value than on account of the facility with which large quantities can be given together with fat. The more butter, cream, etc., a food of this kind can absorb without revealing the taste of fat, the more useful it is in a fattening cure. In this respect Grape Nuts occupy first place.

I cannot endorse the popular recommendation of cocoa as a fattening means, for cocoa rather destroys than stimulates the appetite and it does not equalize this defect by a very high nutritive value. In addition, cocoa frequently predisposes to constipation. Of the preparations of cocoa that I am familiar with, *Rademann's* nutritive cocoa (containing milk sugar) and *Meinl's* Maltine Cocoa form an exception. They stimulate peristalsis and, while possessing higher nutritive value than ordinary cocoa, do not satiate so much. Both permit the abundant addition of butter and cream.

The greatest possible increase in carbohydrates is of particular importance in cases in which it is desired to increase the albumen content of the body; for the albumen sparing influence of the carbohydrates, that has been pointed out in particular by *Voit's* school, is, from a practical standpoint, more important than the retention of albumen that may be forced by an exaggerated increase of the albumen intake.

The following table demonstrates what quantities of different important articles contain 100 g of carbohydrate in addition, of course, to other substances:

Milk	2100-2500 ccm
Sour milk	28 ccm
Oatmeal	150 g
Hulled rice	130 g
Corn meal	140 g
Peas and lentils (dry)	200 g
Bread	180- 200 g
Zwieback and biscuit	120- 135 g
Potatoes	600 g
Macaroni	135 g
Sugar	100 g
Honey	140 g
Fresh fruit (average)	1000 g
Strawberries, raspberries	2000 g
Dry fruit	180- 220 g
Grapes	500- 600 g
Chestnuts (peeled)	130- 140 g
Fruit marmalades	400- 500 g
Grape juice	500- 600 g
Other fruit juices (without the addition of sugar)	700- 800 g
Beer (ordinary)	1800-2000 ccm

Bock beer	1500-1600 ccm
Malt beer	800-1000 ccm
Braunschweiger Mumme	200 g

3. Fat. Unless particular reasons argue against its employment, fat is among all articles of food the most valuable in fattening cures. It owes its position to the property it possesses to a pre-eminent degree of representing a high caloric value (1 g = 9.3 cal.) in a small volume. Individuals who wish to undergo a fattening cure are almost without exception poor fat eaters and have frequently been so from childhood up. If they had not been bad fat eaters, or if they had not become so, it would not have become necessary to fatten them, for any individual who eats sufficient fat possesses at least an average minimum body weight (aside possibly from sufferers from certain diseases like diabetes, exophthalmic goitre, etc.). Frequently the small ingestion of fat is merely a matter of habit. Occasionally there is an aversion to fat, frequently based upon the prejudice that fat is under all circumstances difficult to digest. There are unfortunately many health cranks who judge everything that is offered to them in the way of food from the standpoint of its ready or difficult digestibility. They rarely utilize their personal experience as a criterion. The latter should be reckoned with somewhat, of course, in practice, although one must anticipate a tendency on the part of such individuals to deceive themselves. Generally the prejudice is based upon imagination, upon the dicta

of other persons, so-called family traditions, or upon pseudo-scientific bits of information that have been picked up here and there. With rare exceptions, that must be occasionally conceded, all this is nothing, however, in reality but prejudice.

Individuals who must undergo a fattening cure rarely ingest spontaneously more than 100 g of fat. I base this statement upon the great number of diet calculations that I have made. Under ordinary conditions 100 g of fat constitute a perfectly safe quantity. *Voit's* "Normal Diet" (for 70 kg of body weight with an average amount of labor) incorporated, as is well-known, aside from 118 g of albumen, only 56 g of fat with a maximum of 100 g when the individual was performing very energetic labor; hence there were derived from the fat only 520 to 930 calories. In addition, however, 500 g of carbohydrate were assumed to be present in this diet, representing 2050 calories. *Voit* arrived at this disproportion between fats and carbohydrates, because he performed his investigations among people who were used to an exceptionally large carbohydrate diet occupying an exceptionally large volume; for we mentioned above that 500 g of carbohydrate represent a volume of at least 2500 ccm. One should not base observations intended to be of general application upon individuals with abnormal habits. In cases, however, in which the consumption of carbohydrate food is materially reduced, as in the case of individuals whom we intend to fatten, the sum of 100 g of fat will be just sufficient, when added to the small amount of carbohydrate and the albumen ration, to aid them in maintaining the balance of nutrition.

The question now arises to what extent can fat be utilized in order to raise the value of the food above the level of the maintenance diet. According to my experience, one has no difficulty in increasing the fat ration up to 250 g per diem, so that the fat alone represents the respectable figure of 2325 calories. It is possible to go still higher, but then one must be prepared for occasional digestive disturbances that may retard the progress of the fattening cure for a number of days or (and this is much more to be feared) may frequently discourage the patient. In so far as I consider it advisable to accustom the patients to a diet that they can maintain for a long time, above all things after the termination of the cure proper, I am inclined to abstain from the beginning from an excessive increase of the fat intake. One should usually be content to bring the daily intake up to 200 to 250 g. When this is done the general diet more nearly represents a natural method of feeding, for 200 to 250 g of fat may very easily be concealed in the rest of the food. As soon, however, as the sum of about 250 g is exceeded, the selection, arrangement and preparation of the food must vary from the ordinary diet, and when this occurs difficulties arise that may not become manifest at once, but will certainly appear if this fat diet is continued for a long period of time. Consequently, under this plan, the permanency of the result becomes doubtful just as in the case of too abundant carbohydrate feeding (see above). One should never forget that the fattening cure proper, that is, the increase of the body weight from 6 to 10 kg during a period of systematic treatment extending over two months, is by no means the most important goal. Much more essential is the permanency of the result, and much more important is also the educational value of the fattening cure proper, for by it we attempt to teach the patients how they must eat in order to continue the strengthening process and to remain permanently strong. A fattening cure is a true course of education that these patients require. They must be educated to eat large quantities of fat. As soon as this result is assured, the danger of a relapse to a poor condition of nutrition becomes very slight.

The importance of the educational element in fattening cures cannot be sufficiently emphasized, and for this reason one has been in the habit for a long time of advising fattening cures to be carried out in a hospital or a sanatorium; for in all really difficult cases constant and continuous contact between the physician and the patient becomes necessary. Routine above all things and the following of a definite schedule are to be condemned. In each individual one must study separately how the caloric value of the food can be raised to the desired total, not only for the moment but permanently. Not only must the patient be instructed by the physician, but the physician should also learn from each patient. One of the main criticisms that has been formulated against various sanatoria is the fact that they lay too much emphasis upon the momentary result and not enough upon the educational factor and upon the after effect of the fattening cure.

No other article of food requires so careful an investigation of individual peculiarities in each patient as fat. It is essential to introduce large quantities of fat. One can almost say that to a certain extent the immediate result and by all means the permanent result in any fattening cure depends upon our ability to accustom the patient to take large quantities of fat. This applies particularly to early stages of pulmonary tuberculosis and to the mass of gastric and intestinal troubles that are treated by fattening cures. Late stages of gastric ulcer, of chronic constipation, of gastric and enteric neurasthenia constitute the principal candidates for fattening cures. The introduction of abundant quantities of fat as an established custom in pulmonary tuberculosis harks back to the days when the safety anchor of tuberculosis was sought for in codliver oil. In functional and in many organic diseases of the digestive canal, the introduction of large quantities of fat was only attempted much later. We live in an age that rapidly forgets, consequently it may not be amiss to remind my readers in this place that it was only my investigations in regard to the utilization of the food in gastric diseases and the investigations that I published jointly with *C. Dapper* that blazed the trail in this direction. Now-a-days we are afraid of abundant fat feeding in gastric and intestinal diseases only in cases suffering from true inflammatory processes about the stomach or the upper sections of the intestine, or in cases in which disturbances of the fat absorption are definitely demonstrable. It is clear that the introduction of fat becomes more difficult the more gastrointestinal disturbances predominate in the general disease picture.

As a rule it suffices in fattening cures to administer a definite fixed amount (“eiser nen Bestand”) of fat-containing foods, that must be introduced in some way or another.

Merely as an example, not as a standard, the following combination may be given:

Butter 200 g	containing	160 g	fat; value of the fat	1490	calories
Milk 1 1	“	33 g	“ “ “ “ “	307	“
Cream (thick) 300 g	“	75 g	“ “ “ “ “	698	“
				Total, 2495	fat calories

Together with the proteids and carbohydrates contained in a liter of milk and together with the caloric values incorporated in other foods added to the above, the highest caloric values that can be utilized at all in fattening cures may be reached on this basis. The amount of fat given in the above example is greater than is generally to be recommended (see above). By the addition of 300 cc of cream the fat intake reaches the high value of 268 g and very frequently it becomes necessary to omit the administration of so much cream, for there are many individuals with otherwise perfectly healthy digestive organs that are very susceptible to cream and suffer a considerable loss of appetite as soon as too much cream is given.

A list of articles of food containing much fat and that can be utilized in fattening cures will be found at the end of the first lecture.

4. ALCOHOL. Alcohol differs fundamentally from the other articles of food, albumen, fats, carbohydrates, in so far as it never becomes, either directly or indirectly, an integral part of the organism. The fat of alcohol is to be burned. In process of oxydation it displaces other substances, in particular fat, and prevents their combustion, and it does this in proportion to the caloric value of the two substances. When 9.3 g of alcohol are burned in the body, 7 g of fat less are oxydized than would have been the case if no alcohol had been administered. In other words, alcohol should be a good and useful means of fattening. Unfortunately its toxic properties militate against its extended use. It is unnecessary at the same time to "spill the baby with the bath"; in other words, to withhold small harmless doses, because large doses of alcohol are undoubtedly injurious. One may even occupy the standpoint of total abstinence as a matter of principle on social and ethical grounds and still not deny the therapeutic value of alcohol and hence employ it as a drug. Unfortunately this error is frequently committed by extreme advocates of anti-alcoholism, and objections to the use of alcohol that correspond in no way with

scientific facts are frequently formulated by these people.

In fattening cures the use of alcohol is not essential and it may even occasionally be injurious. Everything depends upon the individual peculiarities of the person one is dealing with. In other cases again, one cannot get along without alcohol, unless one wants to dispense from the beginning with a valuable and under certain conditions determining adjuvant to the course of treatment. The caloric value of the alcohol is a minor consideration, for one will rarely give more than 50 g of alcohol per diem, and this amounts to only 350 calories, a very modest sum that can readily be supplied by other means. Only in severe cases of diabetes in which, to judge from my experience, that is being constantly corroborated, larger quantities of alcohol (up to 100 g a day) play an important and life-saving rôle, does the caloric value of the alcohol administered count. In fattening cures, as a rule, alcohol serves more as a stomachic and a nerve tonic by enabling the patients often, though not invariably, to eat larger quantities of food. It is also a well-known fact, borne out by common experience, that the addition of alcohol to the food aids in the assimilation of the fats. It is too bad that these advantages must be continuously dinned into the ears of temperance advocates.

For the purposes enumerated above very small quantities of alcohol frequently suffice. I prefer wines containing from 15 to 20% of alcohol (so-called dessert wines) given in quantities not to exceed

50 ccm at a time and, in addition, in the evening after the last meal and to end the day with, occasionally a glass of heavy beer containing abundant extractives. A beer of this character, for instance, is Munich Bock beer (Salvator) or English porter. Among the Austrian brands of beer, double malt beer (St. Stefan) is particularly to be recommended. A combination of the extractives of meat with alcohol seems to exercise a particularly favorable influence upon the appetite and hence possesses considerable significance in fattening cures. The refinements of the culinary art have been utilizing this principle for a long time. I mention the addition of Madeira and of other wines to strong meat broths, oxtail soup, mock turtle soup and many sauces. Not only is the taste of these preparations improved thereby, but a stimulating effect upon the appetite is at the same time attained. There are several preparations in the market that utilize this old experience. I mention, for instance, *Vial's* Tonic Wine, that in addition to meat extractives also contains certain bitter tonics. *Erb,* who is otherwise no great advocate of alcohol in nervous diseases, praises the importance of this preparation as an adjuvant to the dietetic treatment of neurasthenic individuals. Basing on a recommendation coming from so eminent an authority, I have frequently used this preparation, and can verify that, while introducing relatively small quantities of alcohol, it decidedly fulfills the above purposes.

V REST AND EXERCISE

When, about 25 years ago, *Weir Mitchell* and *Playfair* introduced a systematic fattening cure as an important means of treatment in hysteria and neurasthenia, rest and isolation were at the same time included in the therapeutic program. We concede nowadays that in numerous cases these accessory means are needed; for the physical condition of the patient frequently demands their employment. In the meantime fattening cures have been introduced in a much wider variety of disorders, so that to-day they are employed not only in the treatment of nervous disorders that are closely related to psychoses, but also in diseases in which the sole aim of the fattening cure is to strengthen the organism as a whole, and in this way to favorably influence and support certain organs whose function has become impaired. Nevertheless, a rest-cure (usually implying several weeks in bed) is generally included both by the laity and by physicians in the idea of a fattening cure; the underlying conception being that the rest of the muscular apparatus by depressing oxydative processes to the lowest conceivable minimum favors a correspondingly increased deposit and storage of the surplus pabulum administered. As a prototype and example the success of farmers in the fattening of cattle and poultry by this means is often quoted.

The underlying principle is correct, but its general application wrong. One can exclude cases that on account of nervous diseases or disorders about other organs (for instance, ulcer of the stomach, tuberculosis of the lungs) require a prolonged rest-cure. Even in the absence of such definite indications one is justified, naturally, in the beginning of any fattening cure in prescribing absolute rest (for a few days to a week) if for no other reason than that it is much easier in this way to accustom the patient to strict regularity in feeding; besides one can under such conditions more readily apply moist, warm or cold abdominal compresses that are often necessary to relieve certain subjective disturbances appearing in persons with hyperesthetic digestive organs. As soon as feasible, however, and as soon as, with the aid of the rest-cure, the first 2 to 3 kg have been gained, muscular exercise should be instituted. Massage is quite useless, for it does not sufficiently strengthen the muscles. Massage may be indicated only in the intermediary transition stage of the treatment. *Active muscular labor* is what is required, for only by this means can the protoplasm-forming forces become mobilized. This point has already been thoroughly discussed (see page 51). It is true that by muscular exercise a part of the flesh that has been gained is sacrificed and that the risk is always incurred of restricting the total gain in weight. As a rule, however—provided muscular exercises are not begun too soon and do not exceed in the total labor performed the actual capacity of the muscular apparatus—a very welcome increase in the appetite will be produced causing a greater ingestion of food that more than overbalances the loss of energy resulting from the physical labor. I have frequently ordered patients undergoing a fattening cure to exercise from one hour from the second week of the treatment on; and beginning with the third week to climb to an elevation each day of from 150 to 250 meters; rowing and swimming exercises are also often instituted. The ultimate gain in weight is by no means less than the gain that would have been accomplished as an optimum had the rest-cure plan been persisted in. Another great advantage is gained hereby, namely, that the patients are not, at the termination of the treatment, merely fat, but actually stronger in muscle development, and find themselves in a good state of nutrition, so that they can resume their daily life with greater confidence in themselves and with an assured and definite working capacity.

From the same standpoint is is advisable to employ *hydrotherapy* as an adjuvant to the treatment as early as possible; the methods to be employed, however, should not be of a softening character, as is so often unfortunately the case, nor should their effect be merely suggestive (radium baths, oxygen baths, irradiation with the different colors of the rainbow, etc.), but really hardening methods that actually have a tendency to exercise the depressed functions back to normal. I cordially agree in this respect with *Winternitz,* who has frequently emphasized these points.

Together with the new-formation of muscle tissue that is forced in this way a *regeneration of the blood* is generally witnessed (see above, page 35). Certain other adjuvants to the treatment may nevertheless be required to fulfill this latter purpose. Thus the patients during their hours of rest should be encouraged to remain out of doors. I have adopted this plan, that has been so popular in the case of consumptives for a long time, in the case of subjects undergoing a simple fattening cure, and with the best results even during the winter months in Germany. Occasionally one cannot get along without certain drugs that are intended to stimulate blood formation. Arsenic, unless one is dealing with chlorosis, by all means deserves the preference over iron; at the same time the exaggerated use of arsenic preparations, in particular the injection of cacodylates that are now-a-days so universally employed, should be energetically condemned. Unless there is present an anaemia due to relaxation of the bone marrow function, such arsenic treatments are essentially without value. We have actually arrived at a place where in simple fattening cures an injection of cacodylate is valued more highly than a beef steak.

In another place (see page 34) reference was made to the fact that the glandular organs must be considered storehouses of protoplasm in the same sense as the muscles and the blood. No particular attention, however, need be bestowed upon these organs during a fattening cure. For with the introduction, digestion and assimilation of more food, the activity of the digestive and excretory glands is automatically increased, and this increased labor exercises and strengthens these parts. The greatest benefits accrue primarily to the gastro-intestinal tract, so that we often witness an improvement in the absorptive and assimilative powers of the digestive organs as the fattening cure progresses. Not only is the glandular apparatus of the digestive tract favorably affected, but the muscle *tonus* and the motor power of the intestine become increased. How often do we witness a disappearance of gastric atony and of sluggishness of the bowel function in the course of a fattening cure. Under certain circumstances, of course, the increased labor that has to be imposed upon certain organs in the course of a fattening cure may force an abandonment of any attempt at fattening, or may necessitate a very particular selection of the diet, as, for instance, in the case of diseased kidneys.

VI INDICATIONS FOR FATTENING CURES

The indications for a fattening cure are exceedingly numerous, and it is not my intention within the narrow frame of these lectures to exhaustively discuss them, for nearly everything that I might say in this respect is already familiar to every practitioner. I will limit myself to a few salient considerations.

1. In Pulmonary Tuberculosis the use of fattening cures was recognized earlier and they were employed more methodically than in any other disease. If we omit the inadequate and incomplete tendencies in this direction that became manifest many years ago, then we must acknowledge our debt primarily to *Brehmer* (Göbersdorf) and to *Dettweiler* (Falkenstein) for having introduced fattening cures into the treatment of pulmonary tuberculosis and for having methodically developed this system of treatment. In the meantime fattening cures have become recognized and valued as one of the most important means at our disposal to combat this disease. The results no doubt are good, although chiefly in early stages of the disease. One must acknowledge unreservedly that a good state of nutrition is necessary to prevent the progress of the disease, and one cannot, therefore, blame the managers of tuberculosis sanatoria if they place the greatest emphasis upon attaining such an improved

state of the general nutrition. Nevertheless, the matter may be overdone. Only too often other no less important factors in phthisiotherapy are sacrificed to efforts directed too exclusively towards attaining an imposing gain in weight. Especially are hardening measures often relegated to the background. One need only compare what has been said by *H. Weber, Brehmer* and *Dettweiler* in their fundamental publications in regard to important factors other than nutrition with what is actually advocated and carried out to-day in order to see the great change that has taken place; hardening factors, it will be found, especially hydrotherapy, are decidedly crowded into the background in most sanatoria. The penalty for this neglect is often paid as soon as the patients return home. Overfeeding in institutions is sometimes carried too far. We would be glad to welcome our patients back from the sanatorium in a good, medium state of nutrition; frequently, however, we are confronted by actually obese individuals, and this is by no means of advantage to the ventilation of the lungs and the working capacity of the heart. I uttered a warning against such exaggerated tendencies a long time ago, and cannot refrain from urgently repeating this warning to-day, since I find myself fortified in my opinions by repeated experiences of this character.

2. BASEDOW'S DISEASE (Exophthalmic Goitre) leads to emaciation because under the influence of hyperthyroidism oxydation is markedly increased. At the height of this disease a sufferer from exophthalmic

goitre consumes from 20 to 50% more energy than a healthy individual under equal conditions. An amount of food, therefore, that would constitute a maintenance diet for a healthy individual under these circumstances naturally leads to undernutrition. For this reason exophthalmic goitre for a large percentage of the patients that enter institutions in order to undergo a fattening cure. One rarely succeeds at home in increasing the food to a satisfactory level; for a sufferer from exophthalmic goitre, as indicated above, requires a very much greater intake of calories than any other kind of a patient in order that he should gain in flesh. There are, in addition, certain other peculiarities about the metabolism of these cases. It is not permissible to give too much albumen, because it stimulates oxydation even more than in healthy individuals (specific dynamic influence of an albuminous diet, *Rubner,* see page No. 42), hence is operative in the same sense as the hyperfunction of the thyroid gland. In addition there is in severe cases a tendency to diarrhoea. If the ejecta are carefully studied, it will be found that as a rule steatorrhoea exists, a sign that the absorption of fats is suffering. Notwithstanding all these contrary factors, it is nevertheless possible to increase the weight of these patients, if they are suitably treated, and notwithstanding the fact that the organism (just as in a fever patient) is naturally averse to accumulating albumen. Not only the accumulation of genuine protoplasm but also the storing of reserve albumen is rendered difficult, so that in these cases fattening cures lead almost alone to an accumulation of fat. Sanatoria point with particular pride to patients suffering from exophthalmic goitre who have accumulated abundant, even over-abundant, fat reserves during their sojourn in the institution. I have personally attempted to obtain such a result and have occasionally succeeded, but I never gained the impression that the patients were very much benefited thereby, for they remained afflicted with exophthalmic goitre, even if they had grown fat. In fact, energetic fattening may be distinctly detrimental to these patients unless it is associated with strengthening of their musculature.

One should never forget that the heart muscle in many patients suffering from exophthalmic goitre is weak and has a tendency to dilatation; as soon as the patients acquire more fat by stuffing and by rest, the heart muscle is subjected to increased exertion, consequently one often finds that these fattened patients are incapable of performing the mildest physical exertion without developing marked acceleration of the heart. In two cases I was a witness to sudden collapse and death from heart failure. One of these cases had been presented to me a few days before his death in a sanatorium as a "cured case of exophthalmic goitre." Although I did not have an opportunity of examining this patient myself, I was informed with pride on the part of the sanatorium management that this patient had gained 12 kg in weight during two months, although prior to the beginning of his treatment he had been very thin and weak. A few days later this patient took a little walk and returned home in a great hurry on account of threatening rain. Immediately after his return he developed marked palpitation, oppression, dyspnoea, cyanosis, thready pulse, and died within half an hour. Such cases, although they naturally only rarely end so badly, should nevertheless constitute a warning.

3. Nervous Dyspepsia. Patients who are reduced on account of nervous digestive disorders of different kinds naturally constitute a large proportion of the individuals who become candidates for a fattening cure. Immaterial whether the difficulty concerns chiefly the stomach or chiefly the intestine, systematic hardening and exercise treatment of the digestive organs must always be combined with the fattening cure proper; hence such cases should be treated in a sanatorium (see above, page 61). Fattening alone accomplishes very little. Unless one succeeds in inducing the patients to take a normal diet and to impose a normal amount of labor upon the stomach and intestine, relapses within a short time are unavoidable. We learn this from daily observation in numerous patients. On the other hand, the results, if everything succeeds, are among the most brilliant achievements of internal medicine. Particularly that numerous class of cases in which the starting point of the trouble is in the intestine (so-called spastic constipation with or without production of mucus and attacks of pain) and in which the stomach is only involved secondarily, as

well as cases of simple gastric and intestinal atony, may become completely cured. The fact that an increased amount of labor is imposed upon the digestive tract constitutes a training for this organ and increases its capacity. Simultaneously with the disappearance of the digestive disorders we witness the rout of a veritable army of nervous disturbances; so that these patients, who up to this time did nothing but complain and groan, are again led back into more pleasant paths of health and strength.

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